Atelectasis and Shunting During Spontaneous Ventilation in Anesthetized Patients

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The hypothesis is tested that progressive atelectasis, with shunting of venous blood into the arterial bloodstream, may occur with ventilation which is normal by the usual criteria, but lacking in periodic deep breaths capable of reinflating collapsed airspaces. A previous study found such shunting to occur in anesthetized patients, ventilated by mechanical respirators; in the present study 25 patients were anesthetized with ether and oxygen and breathed spontaneously for an average period of 130 minutes. At the end of this period of spontaneous ventilation the average arterial oxygen tension was 402 mm. of mercury. Following a period of three to five minutes of controlled ventilation, using large tidal volumes, the average arterial oxygen tension rose to 553 mm. of mercury. The greatest fall in arterial oxygen tension occurred in the patients whose spontaneous tidal volumes were the most shallow. It is concluded that spontaneous ventilation in anesthetized patients, even when adequate in terms of carbon dioxide elimination, should be supplemented with periodic passive hyperinflations.

THE PRESENT study was undertaken to examine whether arterial oxygen tension falls below expected values during spontaneous ventilation, and whether such falls are reversible by hyperinflation of the lungs.

At every return to the resting end-expiratory position some airspaces collapse and stay collapsed during continued tidal ventilation. Such progressive atelectasis persist until a deep breath, active or passive, applies sufficient pressure to reopen the atelectatic areas of the lungs. Mead [1] proposed this interpretation of data, obtained in dogs, showing progressive decrease in pulmonary compliance during quiet breathing, and return of compliance to control values following passive hyperinflation. The role of the periodic deep breath in maintaining normal compliance was demonstrated in conscious humans by Ferris,[2] and in anesthetized patients by Egbert.[3]

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If collapsed aveoli continue to be perfused oxygenation will not take place in these areas of atelectasis, and venous blood is shunted into the arterial bloodstream. In a previous study the authors demonstrated falls in arterial oxygen tension and pulmonary compliance in anesthetized patients during ventilation with mechanical respirators. Reversal was possible by sustained passive hyperinflation of the lungs.[4] In dog experiments Laver examined the value of lung volume, pulmonary compliance and alveolar-arterial oxygen differences as indices of atelectasis; oxygen tension measurements were proposed to be the practical diagnostic tool.[5]

Methods and Procedure

Twenty-five routine surgical cases were chosen for study; an equal number was sought of operations on extremities, and on lower and upper abdomen. The patients had no manifest pulmonary disease and presented no contraindications to general anesthesia with spontaneous ventilation.

Preanesthetic medication consisted of pentobarbital sodium, approximately 1.5 mg. per kilogram, and atropine sulphate 0.6 mg. Anesthesia was induced with thiopental sodium in divided intravenous doses, and endotracheal intubation was facilitated by succinylcholine chloride in a single dose of 40 to 80 mg. One patient had induction with nitrous oxide-ether alone. Spontaneous ventilation was then reestablished and anesthesia maintained with ether-oxygen in a circle rebreathing system.* The anesthetists were instructed not to interfere with spontaneous ventilation, and it was

* Under the conditions of the study total gas flows in the anesthesia circuit (ranging from 3 to 5 liters per minute) cannot significantly influence either the rate of air space collapse or the concentration of oxygen in inspired air, except that the rate of denitrogenation is facilitated by high gas flows. Denitrogenation was assumed to be essentially complete before the first samples were drawn.

TABLE 1. Spontaneous Ventilation

Operations On:	Number and Sex	Duration in Minutes	Age in Years	Weight in Kilograms	Tidal Vol. in ml. per Kilogram Body Weight	Resp. Rate per Minute	Art. Oxygen Tension mm. Hg	Art. Carbon Dioxide Tension mm. Hg	Art. pH Units
Extremities	8 5 males \ X 3 females / SD	131 ±74	61 ±17	73.8 ±12.8	5.00 ±0.74	25.9 ±3.9	403.1 ±152.2	44.2 ±5.2	7.37 ±0.04
Lower abdomen	9 5 males \ X 4 females / SD	153 ±86	53 ±20	61.9 ±12.1	4.77 ±0.84	30.8 ±6.9	399.1 ±134.4	40.2 ±12.1	7.36 ±0.09
Upper abdomen	8 5 males \ X 3 females / SD	103 ±44	59 ±16	69.0 ±11.9	4.13 ±0.82	31.9 ±5.0	404.8 ±135.7	45.9 ±9.0	7.34 ±0.06
Total	25 15 males \ X 10 females / SD	130 ±74	58 ±18	67.9 ±12.8	4.64 ±0.81	29.6 ±5.9	402.2 ±126.7	43.3 ±9.2	7.36 ±0.07

specifically requested that assisted ventilation be omitted completely and that passive hyperinflation not be carried out at any time.

Before closure of the surgical incision, at a time when anesthesia was maintained by very low concentrations of ether in inspired oxygen, the average respiratory rate was counted and the average tidal volume measured by a Wright Respirometer, during a five minute period. Arterial blood was then withdrawn through a Cournand no. 18 needle, indwelling in a radial artery. Each syringe, wet with heparin, was capped immediately and all determinations carried out within ten minutes. Samples and measurements were made in duplicate, all values corrected to 37° C.†

Following the first set of samples the pattern of ventilation was changed by manual control, using a slow rate and large tidal volumes; every breath was intended to be a hyperinflation. After three to five minutes of controlled ventilation, with the pattern of slow, deep breaths maintained, a second set of arterial samples was withdrawn. No change was made in the inspired mixture during this period.

Arterial oxygen tension was measured by means of modified Clark electrodes, which in our laboratory have a reproducibility of ± 2 per cent. Arterial carbon dioxide tension was determined using a Severinghaus electrode (reproducibility ± 2 per cent), and *p*H by a Beckman glass electrode. Amplification and recording were made on a Sanborn no. 350 system.

Two patients were in a modified lateral position, 4 in a slight head-down tilt (less

† The considerations applying to calibration and correction for temperature of the oxygen electrode are presented in detail by Hedley-Whyte and Laver.[6]

TABLE 2. Effect of Deep Breathing

Operations	Arterial Oxygen Tension mm. Hg	Increase in Arterial Oxygen Tension mm. Hg	Arterial Carbon Dioxide Tension mm. Hg	Arterial pH Units
Extremities	545.9 ±127.4	142.8 ±86.5	23.8 ±6.5	7.52 ±0.07
Lower abdomen	572.7 ±90.6	173.6 ±112.9	22.6 ±5.9	7.53 ±0.08
Upper abdomen	537.4 ±81.7	132.6 ±89.6	27.9 ±7.9	7.47 ±0.08
Total	552.8 ±88.1	150.6 ±95.1	24.7 ±6.7	7.51 ±0.08

than 30 degrees), and the rest were supine and flat on the table.

Sustained single hyperinflations occasionally caused falls in arterial blood-pressure. Circulatory recovery occurred promptly, when the intrathoracic pressure was reduced, and this effect was entirely avoidable by limiting the duration of increased airway pressure to a few seconds, and by allowing a prolonged end-expiratory pause at ambient pressure.

Results

Spontaneous ventilation for an average of 130 minutes led to mild respiratory acidosis, and to a mean arterial oxygen tension of 402 mm. of mercury (table 1). The differences between categories of operations were minor and not statistically significant.

The period of deep and slow breathing, manually controlled, caused an average rise (significant, $P < 0.01$) in arterial oxygen tension of 150 mm. of mercury, to a new mean value of 553 mm. of mercury (table 2). Hyperventilation was not intended, but did occur nevertheless, converting the mild respiratory acidosis into a mild respiratory alkalosis.

From patient to patient the arterial tension of oxygen, in spontaneous ventilation, varied considerably, as suggested by the relatively large standard deviations. The relation of arterial oxygen tension to other factors was tested in order to determine what causes the varying degrees of impaired oxygenation during spontaneous ventilation. The tidal volume per kilogram body weight was linearly related to the arterial oxygen tension (fig. 1), suggesting that the greatest impairment of oxygenation will occur when tidal volumes are most shallow.

In this study there was no demonstrable influence of age, sex, duration of operation, site of operation, body weight or respiratory rate on arterial oxygen tension. The few patients not in the supine position represented no apparent influence.

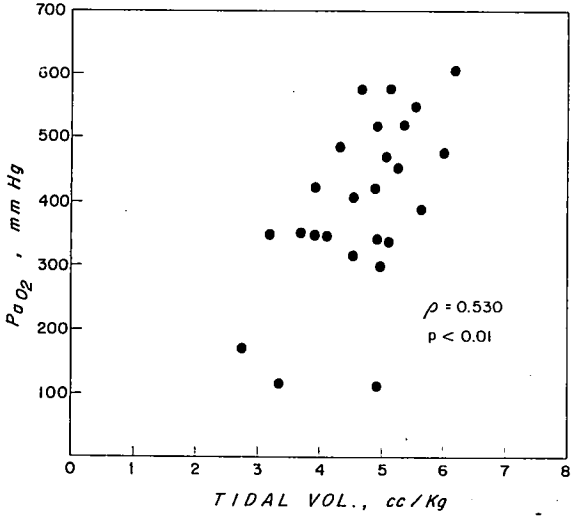

FIG. 1. The Rank correlation (Spearman) of arterial oxygen tension, following spontaneous ventilation, plotted against tidal volume per kilogram body weight is statistically significant (rho = 0.530; $P < 0.01$), suggesting that shallow breathing leads to greater degrees of atelectasis and shunting.

Discussion

Atelectasis with right to left shunting is assumed to cause the low arterial oxygen tensions during spontaneous ventilation. The subsequent rise in oxygen tension, following a series of deep breaths, is the likely result of reopening collapsed airspaces and restoration of a more favorable relationship of ventilation to perfusion.

The relation in this study of the degree of impaired oxygenation to the pattern of ventilation confirms earlier findings by the authors during controlled ventilation in anesthetized patients.[4] In dogs, Hedley-Whyte demonstrated that shallow tidal volumes cause significant falls in arterial oxygen tension, while large tidal volumes effectively maintain normal arterial oxygenation.[7]

If a low arterial oxygen tension (with 100 per cent oxygen in inspired air) was caused by simple hypoventilation, the oxygen tension should have fallen only as many millimeters of mercury as the carbon dioxide tension had risen. A ventilatory pattern of shallow breaths at a fast rate may well give a normal (or even below normal) arterial carbon dioxide tension, but atelectasis will cause a fall in arterial oxygen tension. On the other hand, a pattern of slow and deep breaths may maintain normal arterial tensions of *both* oxygen *and* carbon dioxide, by preventing atelectasis and shunting. We consider the pattern of ventilation the single most important factor in determining the degree of atelectasis and shunting.

A high concentration of oxygen in inspired air (approaching 100 per cent) eliminates unequal distribution of ventilation and impaired diffusion as causes of a gradient between alveolar and arterial oxygen tension.[8] In the patients studied only water vapor, carbon dioxide and small amounts of ether shared the total pressure with oxygen. Even allowing for an anatomical shunt of 2 per cent,[9] an increased alveolar-arterial oxygen gradient in older patients and small amounts of ether, the arterial oxygen tension should range from 600 to 650 mm. of mercury in healthy patients.[10] Under the circumstances of this study an increased physiological shunt is the logical explanation for the oxygen tension averaging only 402 mm. of mercury. The substantial rise following the simple administration of very deep breaths makes atelectasis the likely cause of this shunt. Although substantial, the effect of the deep breaths was not consistently a complete return of oxygen tension to expected normal values. The reason for the incomplete reversal is not known, but the duration of ventilation without effective deep breaths (average: 130 minutes) may have been too long to permit complete reinflation with the airway pressures applied. The optimal interval between hyperinflations is not known, but we suspect that it may be in the range of ten to thirty minutes.

The incidence of postoperative atelectasis is clearly related to the site of operation, with the highest incidence following high abdominal and chest operations.[11] This study finds no such intraoperative influence of the site of operation; a similar conclusion is drawn by Stark.[12]

Ether was chosen deliberately as the anesthetic agent, because it exerts no profound respiratory depression and it permits a high concentration of oxygen to be maintained in surgical anesthesia. Furthermore, ether anesthesia has the reputation of displaying a "sighing" type of respiration, even in surgical anesthesia. A few such sighs were seen during this study, but they appeared abortive and incapable of hyperinflation.

The question is often raised whether spontaneous ventilation in the anesthetized patient is ever adequate. History shows that spontaneous ventilation has been tolerated by untold millions of anesthetized patients. But spontaneous ventilation is rarely adequate if normal blood gas exchange is to be maintained, and if safety of anesthesia is to be improved further. Respiratory acidosis must be prevented, whenever present or suspected, by assisted or controlled ventilation, and atelectasis with consequent falls in compliance and oxygen tension must be limited, either by maintaining large tidal volumes at a slow respiratory rate, or by imitating the pattern of ventilation in normal man[13] by providing periodic passive deep breaths capable of reinflating atelectatic airspaces. Spontaneous ventilation in anesthetized patients would undoubtedly be safer if periodic passive hyperinflations were applied.

Summary

A mean arterial oxygen tension of 402 mm. of mercury was found in twenty-five surgical patients, breathing spontaneously and receiving an inspired mixture of ether in oxygen. Following a succession of very deep breaths the arterial oxygen tension rose an average of 150 mm. of mercury. Atelectasis is suggested as the likely cause of increased physiological shunting during tidal ventilation, lacking in periodic deep breaths.

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References

1. Mead, J., and Collier, C.: Relation of volume history of lungs to respiratory mechanics in anesthetized dogs, J. Appl. Physiol. 14: 669, 1959.
2. Ferris, B. G., Jr., and Pollard, D. S.: Effect of deep and quiet breathing on pulmonary compliance in man, J. Clin. Invest. 39: 143, 1960.
3. Egbert, L. D., Laver, M. B., and Bendixen, H. H.: Intermittent deep breaths and compliance during anesthesia in man, ANESTHESIOLOGY 24: 57, 1963.
4. Bendixen, H. H., Hedley-Whyte, J., and Laver, M. B.: Impaired oxygenation in surgical patients during general anesthesia with controlled ventilation. A concept of atelectasis, New Engl. J. Med. 269: 991, 1963.
5. Laver, M. B., Morgan, J., Bendixen, H. H., and Radford, E. P., Jr.: Lung volume, compliance and arterial oxygen tensions during controlled ventilation, J. Appl. Physiol., In press.
6. Hedley-Whyte, J., and Laver, M. B.: O_2 Solubility in blood and temperature correction factors for Po_2, J. Appl. Physiol., In press.
7. Hedley-Whyte, Bendixen, H. H., and Laver, M. B.: The effect of changes in tidal ventilation upon physiologic shunting, Amer. J. Physiol. 206: 891, 1964.
8. Finley, T. N., Lenfant, C., Haab, P., Piiper, J., and Rahn, H.: Venous admixture in the pulmonary circulation of anesthetized dogs, J. Appl. Physiol. 15: 418, 1960.
9. Aviado, D. M., Daly, M. D., Lee, C. Y., and Schmidt, C. F.: The contribution of the bronchial circulation to the venous admixture in pulmonary venous blood, J. Physiol. 155: 602, 1961.
10. Raine, J. M., and Bishop, J. M.: A-a difference in O_2 tension and physiological deadspace in normal man, J. Appl. Physiol. 18: 284, 1963.
11. Hamilton, W. K.: Atelectasis, pneumothorax and aspiration as postoperative complications, ANESTHESIOLOGY 22: 708, 1961.
12. Stark, D. C. C., and Smith, H.: Pulmonary vascular changes during anaesthesia, Brit. J. Anaesth. 32: 460, 1960.
13. Bendixen, H. H., Smith, G. M., and Mead, J.: The pattern of ventilation in young adults, J. Appl. Physiol. 19: 195, 1964.

HALOTHANE AND BLOOD CLOTTING Thirty patients who underwent halothane anesthesia were examined in respect to the following: determination of antithrombin III; prothrombin time; factors V, VII, VIII, IX; platelet count; heparin; fibrinogen; fragility of red blood cells; and overall clotting time. Only minimal variations of all parameters occurred which consisted of a slight prolongation of the overall clotting time shortly after induction of anesthesia. One hour after the end of anesthesia all modalities approached the preanesthetic values. The slight variations were ascribed to changes in circulatory and pulmonary function that occur during anesthesia with spontaneous respiration. The liver was excluded as a causative factor since changes in liver function (if any) do not occur within the short time of these observations. Halothane does not contribute to the incidence of postoperative bleeding or thrombosis; in fact, halothane seems to be indicated in patients with coagulation defects. (*Müller, K. H., Busse, J., and Oehmig, H.: Does Halothane Anesthesia Contribute to the Occurrence of Postoperative Thromboembolism?, Langenbeck Arch. Klin. Chir. 303: 170, 1963.*)